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ENVIRONMENTAL TOBACCO SMOKE

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OVERVIEW

The issue of the reported association between environmental tobacco smoke (ETS) exposures and chronic diseases in nonsmokers has generated a great deal of scientific interest. The first published studies reporting an association between exposure to ETS and chronic diseases in nonsmokers appeared in 1980 and 1981. One study claimed that nonsmokers who worked in environments with smokers exhibited reduced lung function;[1] and two studies concluded that nonsmoking wives of smokers run a higher risk of developing lung cancer than do nonsmoking wives of nonsmokers.[2,3] Another study suggested that ETS was a major indoor pollutant.[4]

The studies of nonsmoking spouses of smokers were followed by similar studies, the vast majority of which do not support the conclusions of the studies mentioned above. In addition, no other study has adequately supported the claim regarding reduced lung function in the workplace. Indeed, each of the reports received criticism by scientists (See, e.g., pp. 9-10, 24-25). Nevertheless, the studies are cited as "proof" that ETS causes disease in nonsmokers. In 1986, for example, the U.S. Surgeon General wrote that the data on ETS were sufficient to conclude that ETS causes lung cancer in adult nonsmokers and respiratory disease in children, and that separation of smokers and nonsmokers does not minimize exposure of nonsmokers to ETS.[5]

Today, after the publication of literally hundreds of reports on ETS and on the broader issue of indoor air quality, the list of health claims regarding nonsmoker exposure to ETS has grown to include the following:

(1) Exposure to ETS causes lung cancer in nonsmokers;

(2) ETS exposure adversely affects lung function in adult nonsmokers;

(3) ETS is related to a decrease in lung function and to an increase in respiratory disease and symptoms in children whose parents smoke;

(4) ETS exposure increases the risk of heart disease in nonsmokers;

(5) Exposure to ETS may worsen pre-existent lung and heart conditions (e.g., asthmatics);

(6) Some people are allergic to ETS, and;

(7) ETS is the major contributor to indoor air pollution.

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The critical question is whether or not such claims are justified on the basis of the scientific literature. A fair reading of that literature shows that none of those claims is convincingly supported by scientific data. In fact, some published studies do not support the claims at all. This conclusion has been endorsed by many physicians, scientists and researchers from around the world. For example, in October, 1986, an international gathering of scientists in Essen, Germany, considered the experimental and toxicological findings regarding ETS.[6] Professor J.G. Gostomzyk, director of the Health Bureau in Augsburg, Germany, concluded in his review of the proceedings that "so far, even toxicology has not been able to ascertain with any greater degree of probability than did epidemiology that there exists a link between damage to health and passive smoking."[7]

In May of 1988, organizers of a symposium on ETS in Austria entitled "Illness Due to Passive Smoking?" issued a press release in which they concluded that "a causal relationship between ETS and illness cannot be established" and that "there is no positive evidence that cancer and other such illnesses are caused by passive smoking."[8]

In his introduction to the proceedings of a symposium on indoor air quality held in Bariloche, Argentina in December 1988, Dr. Osvaldo Fustinoni, vice-president of the National Academy of

Sciences in Buenos Aires, wrote that because of the uncertainty regarding scientific data on ETS exposures and health effects, "it is in question to what extent governments should establish regulations affecting individual freedom, legislating over private facilities like factories or cinemas, theatres, etc."[9]

In his "Summary and Concluding Remarks" from the Proceedings of the International Symposium on ETS at McGill University 1989, (Montreal, Canada), co-organizer Dr. Joseph Wu concluded that the published data on ETS, when critically examined and evaluated, do not provide a scientific justification for the claim that ETS is a cause of disease in nonsmokers.[10]

Dr. Hitoshi Kasuga, in the Proceedings of the International Conference on Indoor Air Quality in Tokyo, Japan, published in 1990, concluded:

> Most participants were of the opinion that it would be very difficult at the present time to reasonably establish a correlation between passive smoking and lung cancer because a detailed examination of all published data (statistical bias in relationship between ETS and lung cancer, estimated ETS levels, incidence of lung cancer in nonsmokers, histological types, etc.) shows that the relationship, if one exists at all, is very slight. Others, however, strongly asserted that there is a correlation.[11]

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ENVIRONMENTAL TOBACCO SMOKE AND HEALTH

Introduction

The claim that exposure to environmental tobacco smoke (ETS) causes chronic diseases in nonsmokers also has generated a great deal of public concern. Although a review of the scientific literature indicates that this claim has not been proved, the literature has been selectively used by antismokers in their efforts to make smoking socially unacceptable and, thereby, to create a "smoke-free" society. The ETS/health issue is so critical to the antismoking movement that the Director of the U.S. Office of Smoking and Health has remarked that "of all the issues, this [ETS] is the one that will propel the United States toward a smoke-free society."[12]

According to one sociologist, the program for a "smoke-free" society may be characterized as follows:

> The strategy, quite overtly, is to progressively stigmatize smoking, segregating the smoker in all public places, and eventually to eliminate smoking as a socially acceptable custom. How is this to be made politically palatable? The answer is clear: by suggesting that smoking harms not only the smoker, but various categories of "innocent bystanders."[13]

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The questionable and often contradictory nature of studies concerning ETS exposure and health is largely ignored by the antismoker; the goal is to achieve publicity and arouse emotion. This strategy is typified by a statement from a commentator on the proceedings of the Fifth World Conference on Smoking and Health: "Regardless of the ultimate validity of the findings, which remains to be established, studies such as these have brought the issue of passive smoking to the public's attention."[14]

In addition, the issue of nonsmoker exposure to ETS has become the means by which antismokers seek to enact legislation regulating smoking in the workplace and other public places. Here again, the strategy of using the ETS/health argument to modify smoking behavior is apparent. For example, the chairperson of the U.S. National Academy of Sciences committee which authored a 1986 report on ETS remarked that "legislation could also be justified, not because it will have much effect on the occurrence of lung cancer among nonsmokers, but because it may motivate some people either to stop smoking or never to start."[15] Indeed, one reviewer observed that there are even instances in the scientific literature in which calls are made to establish ETS as harmful "as a means to limit smoking."[16]

However, the tactic of using the ETS/health argument to modify smoking behavior has been criticized in the international

press. One columnist for The (London) Times noted that while she agreed in principle with the antismoking movement, she could not condone "the lying that accompanies it." She explained: "The lie is the claim that the health hazards of second-hand smoke have been scientifically established."[17] Similarly, a writer for a German publication called the use of the ETS/health argument an "unethical" tactic intended to instill "anxiety in the nonsmoker" and "feelings of guilt in the smoker."[18]

Certainly, ignorance of scientific procedures and the inability to recognize faulty research methodology also play a role in the controversy on ETS. As one reviewer of the literature wrote in 1987:[19]

> A pluralistic society such as ours has lobbies. And it is they who have removed the subject of passive smoking from the scientific to the political stage. It would be wrong to impute this to bad intentions. Inadequate or biassed occupation with the problem, or general insecurity with regard to scientific comprehension, are quite enough.

Given the importance of the ETS issue for the antismoking movement, the purpose of this paper is to review the current scientific literature regarding environmental tobacco smoke and disease in nonsmokers. The intention is to identify sources of information and misinformation about ETS in an effort to demonstrate that the weight of the scientific evidence does not justify the claim that ETS causes disease in nonsmokers.

Lung Cancer

Two highly publicized articles published in 1981 are still among the most frequently cited to support the claim that ETS exposure increases the nonsmoker's risk of lung cancer. A study of Japanese women by Dr. Takeshi Hirayama reported that nonsmoking wives of heavy smokers have a much greater risk of developing lung cancer than nonsmoking wives of nonsmokers.[3] In a study of Greek women, Trichopoulos, et al., concluded that a nonsmoking woman whose husband smokes has twice the risk of developing lung cancer as a nonsmoking woman married to a nonsmoker.[2]

Both studies have been widely criticized in the scientific literature. Numerous inadequacies and inconsistencies in the Hirayama study have been pointed out by noted scientists and physicians who have questioned the validity of its conclusions.[20-41] Dr. Ragnar Rylander, the organizer of the 1983 University of Geneva symposium on ETS, noted that the study had been criticized for its lack of questionnaire reliability, absence of histological diagnosis, questionable statistical treatment, and failure to examine such factors as air pollution from heating and cooking.[41] A participant at the International Conference on Indoor Air Quality held in Tokyo in 1987 observed that Hirayama's study had been questioned for bias in statistical processing, as well as for

failures to measure ETS exposure levels and to evaluate and report "dietary and occupational factors known to influence susceptibility to lung cancer."[22] Moreover, two studies presented at the Conference on Indoor and Ambient Air Quality, held at London's Imperial College in 1988, reported that Hirayama had not divided his study population into appropriate age groups. The presenters noted that the risk he reported for nonsmoking wives of smokers disappears when the age of the subjects is treated properly.[21,27]

Criticisms of the Trichopoulos, et al., study were acknowledged by the authors themselves in an update of their study.[42] They noted that their research had been "criticized (by ourselves and others) because of the small number of subjects, because several tumors lacked histological confirmation, and because controls and cases were from different hospitals."

Moreover, their findings were inconsistent with another study published that same year. In late 1981, Lawrence Garfinkel, an official with the American Cancer Society (ACS), reported the results from a follow-up of the group's long-term lifestyle study, involving nearly 180,000 nonsmoking American women divided into categories based on the amount their husbands smoked.[43] By comparing the lung cancer mortality rates of women reportedly exposed to different levels of tobacco smoke, the differences observed were not statistically significant and that "compared to

nonsmoking women married to nonsmoking husbands, nonsmokers married to smoking husbands showed very little, if any, increased risk of lung cancer." Moreover, Garfinkel cautioned that any study based solely upon classifications of the husbands' smoking habits could not account for total exposure, and that, therefore, the results could be misleading. (See also Garfinkel et al., 1985.)

Several subsequent studies reported results which some claim to support the conclusions of Hirayama and Trichopoulos, et al.,[44-49,51] but a close examination of the methods and conclusions of these studies reveals that their claims are scientifically unfounded. For example, a report by Knoth, et al., which identified 39 nonsmoker lung cancer cases, asserted that exposure to ETS was the "most plausible explanation" for the reported lung cancers.[49] However, the study failed to provide a comparison group as a control population. One reviewer consequently characterized the report as containing "only tentative conclusions based on poor data analyzed by unacceptable methods."[50]

In a 1983 study, Correa, et al., reported an increased risk of lung cancer for nonsmokers married to smokers in a southern U.S. state.[51] However, the results were based on a small number of cases and did not take into account occupational, indoor and outdoor exposures or other confounding variables. Furthermore, one of the co-authors of this study later issued a report with

contradictory findings for a similar population in a neighboring state.[52]

The complex nature of the scientific data in this area is further illustrated by the mixed results of some studies; that is, certain data in a study may suggest a relationship between ETS and lung cancer while other data from the same study may not. For example, Kabat and Wynder, in a 1984 study of 25 male and 53 female nonsmoker lung cancer cases, reported a greater amount of ETS exposure at work for male nonsmoking cases.[53] However, no significant effect was reported for males exposed to ETS at home or for female cases exposed either at work or at home.

In 1985, Garfinkel, et al., published a study of 134 nonsmoking women with lung cancer who were selected from hospital records from 1971 to 1981.[54] Garfinkel, et al., provided two analyses of the same data which produced apparently contradictory results. While a statistically significant dose-response relationship was reported between nonsmoking female lung cancer patients and the number of cigarettes smoked per day by their spouses, no significant relationship was observed between the occurrence of lung cancer in nonsmoking women and the total number of hours per day of exposure to ETS over either the past five or 25 years.

Japanese researchers in 1988 reported small elevated risks for lung cancer among nonsmokers whose parents smoked, but "no association was observed" among nonsmoking wives of smokers who reported ETS exposures either at home or at work.[55]

In another 1988 case-control study of Chinese women, researchers reported a small but significant association between lung cancer in nonsmoking women and spousal smoking. No significant associations were reported between lung cancer and exposure to ETS from parents or colleagues.[56]

Inoue and Hirayama (1988) reported the results of a case-control study on lung cancer in nonsmoking women from two cities in Japan. The authors reported a marginally significant increased lung cancer risk among nonsmoking women whose husband's smoked more than 20 cigarettes per day. However, the data revealed no significant associations for lung cancer in relation to spousal smoking of less than 19 cigarettes per day, or for the overall category of "being married to a smoker" (both exposure categories combined).[57]

In a recent highly publicized article, researchers from the U.S. reported that high household exposures to ETS (e.g. two packs a day for many years) during childhood and adolescence could double the risk of lung cancer. However, the study noted that

exposures to spousal smoking, smoking in the workplace, and smoking in social settings were not significantly associated with nonsmoker lung cancer incidence, nor were cumulative exposures during adulthood or over a lifetime.[58]

Still other studies have observed no significant association between ETS and lung cancer. These include a 1982 study from Hong Kong which reported fewer "passive smokers" among lung cancer patients than among controls.[59] The authors noted that "this finding is at variance with that of Dr. Hirayama's."

Similarly, Koo, et al., studied 200 female lung cancer cases among Chinese women and concluded that ETS exposure in the home or at work had no statistically significant impact on lung cancer incidence.[60] In a second study published in 1987, Koo et al., calculated total ETS exposures at home and at work for their population of nonsmoking female patients. They observed no significant disease trends for any of the lifetime measurements of exposure to ETS.[61]

In two papers published in 1988, Dr. Koo re-analyzed her data on nonsmoking wives of smokers.[62,63] Her results indicated that wives of nonsmoking husbands had "healthier" lifestyles than wives with smoking husbands; they exhibited higher socio-economic status and had better indices of family cohesiveness and lower

frequencies of selected health problems and complaints. An important and statistically significant difference was found in the diets of the two groups. Wives with smoking husbands consumed more processed and spicy foods and ate fewer fresh fruits and vegetables than did wives of nonsmoking men. Koo concluded that such correlates of smoking status "act as important confounders when evaluating health risks among families with smoking husbands."

In 1985, Wu, et al., reported on data from a study of female lung cancer cases in a large metropolitan area in the U.S.[64] They reported that the lung cancer risk among the 31 nonsmokers in the study population was not significantly affected by ETS exposure.

In 1986, researchers investigated the potential role of ETS on the incidence of nonsmoker lung cancer in England.[65] The researchers reported no significant trends in increased risk for lung cancer patients who reported exposure to ETS. In fact, in some disease categories, more controls than cases reported exposure to ETS.

More recently, preliminary results from the on-going American Health Foundation study of 90 nonsmoker lung cancer cases indicate no statistically significant associations for lung cancer among males or females who were exposed to ETS during childhood or adulthood (in the home or the workplace).[66]

Sobue and colleagues, in a 1990 study of lung cancer among nonsmoking women in Osaka, Japan, reported no statistically significant associations between ETS exposures during childhood or adulthood and lung cancer among nonsmokers. The researchers noted, however, a statistically significant increased risk of lung cancer among women who had used wood or straw as cooking fuels.[67]

In 1989, Swedish researchers assessed the relationship between ETS exposure and lung cancer among 38 never-smoking women. They reported no statistically significant associations between lung cancer incidence and ETS exposures in childhood, adulthood or over a lifetime.[68]

Most recently, a joint Chinese and American study reported:

> The result shows that, in females who do not smoke, the presence of lung cancer is statistically significantly associated with chronic bronchitis and family history of lung cancer, the results also suggest an association of lung cancer with duration of cooking food, but not with passive smoking.[69]

Perhaps as a result of the failure of individual studies to report consistently significant results, certain researchers

have recently begun using another method of statistical analysis which combines the reported results from numerous studies. With this method, called "meta-analysis," they claim to have calculated estimated excess risks for nonsmokers exposed to ETS that are 10 to 50 percent greater than those for nonexposed nonsmokers. In basic terms, they have reached these broad conclusions or "generalizations" by calculating an average of the relative risks for nonsmoker lung cancer cases reported by those studies.[70-72] However, this method of generalization has been criticized for its questionable application to the epidemiological studies on ETS, due to the wide variation in their study designs, population selections, techniques of analysis and results.[73-75] This method also ignores basic methodologic weaknesses which, according to one recent governmental report, are characteristic of each published study on ETS and lung cancer.[76] It further ignores the fact that the majority of studies considered in the various "meta-analyses" do not report statistically significant increases in lung cancer risks for nonsmokers. For example, only two of the thirteen studies considered by the 1986 NAS Report achieved statistical significance.[77] (Although the authors of several studies report risks which are not statistically significant, they report "positive" trends in their data. However, trends which fail the test for statistical significance do not reject the hypothesis that there is no relationship between ETS exposures and lung cancer). Indeed, of the nearly 30 studies on ETS and lung cancer

in nonsmokers published to date, only five of the total can claim statistical significance, and the reported elevations of risk are small.[78]

In a recent analysis, Letzel and co-workers assessed the epidemiological studies on ETS for scientific rigor and then performed a meta-analysis on the data pooled from those studies.[74] Of the many possible combinations in the ten case-control studies examined by the researchers, less than 3% gave rise to statistically significant results. Moreover, the findings of statistical significance were explained by the investigators as due to the presence of three studies of questionable scientific merit.[79] The authors therefore concluded that any computed excess risk would be negligible and could not be used to support the claim that ETS exposures increase the risk of lung cancer in nonsmokers.

Peter Lee, a statistician and epidemiologist from the United Kingdom, has argued that the increased risks reported in various "meta-analyses" are the result of an inherent bias in study design rather than the result of any genuine effect from exposure to ETS.[80-85] Lee presents data which indicate that the reported risks cannot be explained on the basis of either ETS exposure or dose for the nonsmoker. It is Lee's contention that the reported "risks" are the result of bias caused by a small number of smokers who are misreported in the studies as nonsmokers.

Other kinds of misclassification may contribute to the reported increase in lung cancer risks among nonsmokers, according to several scientists. For example, none of the studies on ETS and lung cancer provides direct observational information on ETS exposures. Instead, spouses, next-of-kin or friends are asked to estimate the amount of ETS to which they think the subject was exposed. Such estimates may lead to a kind of misclassification, called exposure misclassification,[86] which has been shown by Garfinkel,[54] Friedman[87] and others[88-91] to lead to improper indices of exposure and incorrect estimations of risk. In Garfinkel's study, for example, relative risks varied from 0.83 and 0.77 when the woman with lung cancer or the husband was the respondent, to a risk of 3.57 when a son or daughter responded.[75] That means that the reported risk for lung cancer in the women exposed to ETS was less than for women not exposed when either the women's or their husbands' estimates were used.

Dr. S. James Kilpatrick, a biostatistician from the Medical College of Virginia, has analyzed another form of misclassification which results "from the tendency of respondents to inflate the amount of ETS exposure for lung cancer cases and deflate the report of exposure for controls."[86] Similarly, Dr. Ernst Wynder, President of the American Health Foundation, notes that "relatives of a nonsmoking lung cancer patient are more likely to report

passive inhalation exposure on the part of their relative than are relatives of a control patient."[92]

A more subtle form of potential bias is known as "publication bias", which stems from the apparent failure by journals to publish studies which report negative or weakly positive results.[93,94] Scientists have recently expressed concern over the growing trend among such journals to overemphasize (and hence to publish) only those studies which report positive increases in risk.[95,96] Published studies which are combined for meta-analyses therefore may not include all investigations on the issue of ETS exposures and lung cancer.

Most of the epidemiological studies on ETS and lung cancer have failed to consider age differences, diet, occupation and exposures to indoor or outdoor pollution as potential confounding elements. The importance of such factors is underscored by seven recently published reports, two from Japan and five from China.[67,69,97-101] The reports suggest that indoor pollution generated by kerosene heaters, coal stoves, liquified petroleum gas and exposures to cooking oil vapors may be responsible for the increased risk of lung cancer among Oriental women. Moreover, in 1989, researchers in the U.S. reported that nonsmokers living with smokers consumed less carotene (Vitamin A) than did nonsmokers who lived with other nonsmokers. They concluded that "dietary beta-

carotene intake is a potential confounder and should be measured whenever possible in studies of the relation between passive smoking and lung cancer."[102] Dr. Ragnar Rylander recently summarized the situation thus:

> Studies evaluating the hypothesis of a relation between exposure to environmental tobacco smoke and lung cancer must take into account other environmental risk or protection factors and the possibility that exposure to environmental tobacco smoke may be confounded. This has not been considered in the majority of such studies. Until this has been done, the claim of causality between environmental tobacco smoke and lung cancer remains uncertain.[103]

In addition, relatively few studies have considered ETS exposures outside the home; only five studies sought information about <u>total</u> exposure to ETS from various sources.[54,60,61,65,104] Interestingly, <u>none</u> of these studies reports a significant association between total ETS exposure and lung cancer.

In addition, eight of the published studies also examined workplace exposure to ETS and the incidence of lung cancer in nonsmokers.[53-55,58,61,64,65,68] Not one of the studies provides adequate support for an association between ETS in the workplace and lung cancer.

Nine of the published studies examined the relationship between childhood exposure to ETS and the subsequent development of lung cancer in adult nonsmokers.[46,51,54,58,60,64,67,68,100] Although one recent study reported an significant increased risk from such exposure, the authors "could not explain" their findings.[58] Data from eight earlier studies fail to reject the null hypothesis regarding childhood exposure and lung cancer in nonsmokers. (This means that they did not attribute an increased risk to ETS.) As Drs. Ernst Wynder and Geoffrey Kabat recently noted: "No consistent association has been reported for lung cancer and exposure to ETS in childhood, which might be expected to exert a greater effect, especially when followed by exposure throughout adulthood."[105]

Dr. Maxwell Layard recently summarized the status of epidemiologic studies on ETS and lung cancer:

> The weak and inconsistent associations seen in the epidemiologic studies of ETS and lung cancer, the fact that bias and confounding cannot be ruled out, and questions about the reliability of the reported results, all indicate that these data do not support a judgment of a causal relationship between exposure to ETS and lung cancer.[78]

Dr. Karl Uberla of Germany explained why any attempts to generalize about the significance of reported results of epidemiological studies on ETS and nonsmoker lung cancer will likely remain unconvincing, due to scientific deficiencies in each of the studies.[106] He wrote:

> The majority of criteria for a causal connection are not fulfilled. There is no consistency, there is a weak association, there is no specificity, the dose-effect relation can be viewed controversially, bias and confounding are not adequately excluded, there is no intervention study, significance is only present under special conditions and the biologic plausibility can be judged to be controversial.

Given these difficulties in interpretation, it is therefore not surprising that an eminent statistician should conclude that "it is unlikely that any epidemiological study has been, or can be, conducted which could permit establishing that the risk of lung cancer has been raised by passive smoking. Whether or not the risk is raised remains to be taken as a matter of faith according to one's choice."[93]

Lung Function/Disease: Adults

The issue of ETS and respiratory disease other than cancer in nonsmokers was raised in a 1980 study by White and Froeb.[1] They measured the small-airways function of smokers and nonsmokers and concluded that nonsmokers exposed to tobacco smoke at work for 20 or more years had reduced function of the small airways compared to nonsmokers not so exposed. Similarly, in 1983, French researchers reported that nonsmoking spouses over 40 years of age who were married to smokers exhibited decreases in pulmonary function compared to nonsmoking spouses of nonsmokers.[107]

The White and Froeb study has been criticized for a number of reasons.[108-111] One scientist, Dr. Michael Lebowitz, criticized the study as follows:

> Even with a biased population, poor study design, and incorrect statistical evaluation, there were no clear-cut, consistent, medically meaningful differences between passive smokers and groups of nonsmokers; a corrected statistical analysis strengthened this conclusion.[112]

Dr. Lebowitz also questioned the finding of the 1983 French study which reportedly showed significant differences in lung function in one part of the study population, although no significant differences between exposed and nonexposed nonsmokers were reported in the population as a whole. Dr. Lebowitz noted

that since the "healthiest" population in the study lived in the most polluted areas, the study may have been flawed due to biased population selection and testing or other confounding factors.

In addition, the White and Froeb and the French studies are not consistent with other research on lung disease and lung function in nonsmokers. For example, a 1984 study of 1,351 German office workers reportedly found no effect on pulmonary function among exposed nonsmokers.[113] In a 1988 update of the study, the investigators noted that "there is no evidence that average everyday passive smoke exposure in the office or at home leads to an essential reduction of lung function in healthy adults."[114] Canadian researchers reported that a group of healthy young adult nonsmokers showed "no consistent reaction" of static lung volumes and insignificant changes of other lung function measurements when exposed to tobacco smoke.[115] Numerous studies of individuals exposed to ETS in the home also present conclusions which are contrary to the White and Froeb and the French studies.[116-121]

Some scientists believe asthmatics to be particularly vulnerable to various environmental influences, including ETS. A 1981 study by Dahms, et al., for example, reported decreases in the pulmonary function of several asthmatics exposed to environmental tobacco smoke.[122] However, the study suffers from several obvious limitations. The subjects, for example, were

challenged under the unrealistic experimental conditions of exposure to high levels of smoke in an enclosed smog chamber. In addition, as the authors themselves noted, their experiment lacked proper controls and the effects observed may have been due to psychological, not physical, factors.

Contrary to the reported findings of Dahms, et al., a Canadian group who examined the reactions of asthmatics to levels of tobacco smoke typically found in public places observed no systematic lung changes after such exposure.[123] Later research by the co-authors of this study provides further support for this conclusion. They reported that respiratory data collected from a group of asthmatic volunteers exposed to tobacco smoke "do not suggest that asthmatic subjects have an unusual sensitivity" to such exposure.[124]

Another report on results from a large-scale epidemiological study in the U.S. suggests that environmental tobacco smoke in the home does not affect symptoms or pulmonary function in either children or adult asthmatics, although dust and pollen in the home apparently can provoke such effects.[125]

Tulane University scientists recently assessed the effects of heavy exposure to ETS in a group of self-reported "tobacco smoke-sensitive" asthmatics. Two thirds of the subjects did not

experience significant changes in pulmonary function even after heavy, prolonged exposure to ETS. Moreover, there was no association between reactions to ETS and hypersensitivity to tobacco leaf extract (a substance commonly used in allergy testing).[126]

A group of researchers from Yale University, in another published study on this issue, examined the short-term effects of ETS exposure on a group of young asthmatic patients.[127] They observed no changes in lung flow rates and concluded that such exposures present "no acute respiratory risk" to asthmatics.

Heart Disease

Epidemiologic studies assessing ETS and the risk of cardiovascular disease do not provide a clear picture. Although three studies reported a small, but statistically significant, increase in heart disease risk among nonsmoking wives of smokers,[128-130] another reported both statistically significant and insignificant results.[131] Two other studies found no significant association for heart disease and ETS exposures.[65,132]

One reviewer, commenting on a recent study on ETS and heart disease risk among nonsmoking Scottish residents,[130] noted that the "evidence on environmental tobacco smoke and heart disease has previously been reviewed and considered inconclusive".[133] He added that the new data in the Scottish study "should not materially affect this view."

In 1984, two German researchers reviewed the available studies on the roles of carbon monoxide (CO), nicotine and other tobacco smoke components in the possible etiology of coronary heart disease in nonsmokers. The authors concluded that "there is little evidence" to suggest that substances found in ETS "may adversely affect the cardiovascular system." They also stated that "neither CO nor nicotine is likely to play a role in the development and progression of coronary heart disease in those concentrations

normally found in passive smokers."[134] In addition, scientists and physicians who examined the issue of carbon monoxide at the 1983 University of Geneva Symposium on ETS agreed that "carbon monoxide from environmental tobacco smoke is not important from a health point of view."

The issue of ETS exposure for individuals with preexisting heart disease arises primarily from a 1978 study by Aronow which concluded, on the basis of examining ten heart patients, that such exposure hastens the onset of heart pain during exercise.[135] However, the Aronow experiment has been repeatedly and severely criticized by a number of authorities, including the U.S. Surgeon General.[136] After reviewing the Aronow study, a Canadian professor of medicine who otherwise maintains that tobacco smoke exposure may be harmful wrote that although the patients showed some increase in carboxyhemoglobin (COHb) or CO blood levels, "the endpoint (the reported onset of anginal pain) is necessarily subjective."[137] He explained, "it is difficult to imagine that enclosure in a very smoky room did not have some emotional impact upon patients who were liable to angina, and the psychological disturbance may have done more to hasten the onset of symptoms than the increase of blood COHb."

In addition, other researchers in the area have been unable to replicate Aronow's reported findings. For example,

researchers from Great Britain measured objective responses of a group of heart patients to smoking and carbon monoxide exposures and found no adverse physical response to any level of exposure.[138]

Other serious questions have been raised regarding the underlying validity of Aronow's work. In 1983, after one U.S. governmental agency reported that Aronow had falsified data in a study it had sponsored, another agency, the Environmental Protection Agency (EPA), conducted an independent review of several of Aronow's studies, including his work on heart disease and CO. The EPA concluded that, because of several irregularities in the study, it could no longer rely on his research to formulate environmental standards.[139-140]

Lung Function/Disease: Children

Perhaps no claim regarding ETS is as capable of provoking strong feelings as the charge that parents who smoke may harm the health of their children. While the issue of parental smoking is laden with emotion, the scientific basis for the claim is difficult to interpret. For example, while one study examines respiratory symptoms or illness such as coughs and colds by questionnaire responses from parents,[141] another measures lung function with special equipment at a school or health facility.[142] In the United States alone, according to one report, this has led to a situation in which studies of ETS and the respiratory health of children are being "carried out by at least three different groups, are employing different populations and methodologies and have led to varying conclusions."[178]

Such studies, each with a different sample size, data collection method and analysis, tend to yield factually incompatible and contrary conclusions. For instance, although certain studies have reported adverse findings,[143-164] others have observed no significant relationship between parental smoking and respiratory illness in children.[119,141,165-176] After a five-year study of over 400 children, for example, Dutch researchers concluded there was "no evidence" that parental smoking had an appreciable effect on respiratory symptoms in school children.[171] A similar conclusion

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was reached by a group of U.S. researchers, including a critic of smoking, who found "no significant relation" between parental smoking and respiratory symptoms in a study of nearly 400 families with 816 children in three cities.[119]

Moreover, the authors of studies reporting adverse effects from ETS exposures among children concede that their conclusions must be viewed with caution because of numerous confounding factors. For example, one group of British researchers acknowledged the possible influence of factors such as cross-infection in the home and genetic susceptibility to childhood respiratory illness and symptoms.[147, 148] More recently, researchers in Hong Kong reported "a highly significant correlation between the frequency of respiratory illnesses" of mothers and their children.[177]

Others have conceded that the reliance on questionnaires for information about respiratory symptoms casts doubt on the findings. In one study which reported a significant association between parental smoking and respiratory symptoms, for example, it was noted that even "slight changes" in the way the questions were phrased could result "in substantial differences in the type of responses one obtains."[150] Similarly, another study observed that there was a significant difference in the respiratory symptoms reported depending on which parent completed the questionnaire.[160]

The importance of such confounding factors was given special consideration in the report from the workshop on ETS sponsored by the U.S. National Institutes of Health.[178] Among the many factors listed in the report were types of heating used, socio-economic status and other variables affecting household conditions, including the number of residents, and demographic and medical characteristics of the study population, such as age, parental symptoms and annoyance responses. The report cautioned "that any study which ignores them will be seriously flawed." The relevance of such factors in affecting the outcome of research on parental smoking is supported by a number of reports which have shown that the use of gas stoves in the home may be independently associated with childhood respiratory disease.[125, 168, 179-182]

In 1988, investigators re-examined thirty studies on ETS exposures among children and evaluated the studies for their scientific validity.[183] They noted that while several studies had reported a statistically significant relationship between ETS exposures and respiratory illness in children, "most studies had significant design problems that prevent reliance on their conclusions." The authors concluded that "many questions remain, and future studies should consider important methodological standards to determine more accurately the effect of passive smoking on child health."

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Other confounding factors independent of parental smoking have been reported recently in the medical literature. For example, studies in the United Kingdom have identified damp housing[174,184-186] and father's occupation[187] as potential explanatory factors for the occurrence of respiratory illness in children. Other recent studies have identified outdoor air pollution,[188-190] infections transmitted during day-care attendance[191] and the use of kerosene heaters and woodburning stoves in the home[192-193] as risk factors for childhood respiratory disease.

The relevance of dampness in the etiology of respiratory symptoms in children is supported by current research which links dampness with the presence of molds, dust mites, fungi and other allergenic microbes. The growth of fungi and molds in the home is directly related to respiratory symptoms and sensitization reactions in some individuals.[186,194-196] A recent investigation reports, moreover, that exposure to ETS does not increase sensitization to common allergens in children.[197]

The contradictory nature of findings on the issue of parental smoking is also apparent in the growing number of studies examining the relationship between parental smoking and children's lung function. Although several reports have claimed that parental smoking results in decreased lung function in children,[144,149,150,198-206] others have not,[207-210] including those of a

group of researchers who have published a series of studies on this subject.[118,125,142,211] In 1982, for example, the latter research group showed that a comparison of body size with lung function eliminated any reported correlation between parental smoking and decreased lung function in their children.[141] Two years later, a reanalysis of families from their study group again showed that "parental smoking did not have a significant effect on children's pulmonary function; smoking habits of others in the household (predominantly siblings) did not have any effect either."[118]

It also has been suggested that exposure to ETS may affect infant birthweight. A 1986 study by Rubin et al., for example, reported lower birthweights among infants with smoking fathers compared to those with nonsmoking fathers.[212] However, the discrepencies between the birth weights were described as "extraordinarily large" by one reviewer. In other words, the results were unbelievable. This apparently resulted because the authors failed to control for important confounding variables such as social class.[213] Moreover, a study published in 1987 reported results which contradicted those reported by Rubin, et al. The authors, MacArthur and Knox, observed that the average weight of infants with smoking fathers was greater than that of infants with non-smoking fathers.[214] Still other studies have reported no significant association between paternal smoking and lower

birthweight among newborn infants.[215,216]

Thus, claims that parental smoking plays a causal role in the development of respiratory symptoms, lower birthweight and reduced lung function in children are not scientifically justified. Such claims are typically based upon a single study of a selected symptom (such as cough or wheeze). These kinds of studies invariably fail to consider nutrition, health habits of the family or other lifestyle variables. Similarly, studies which report reduced lung function in children of smoking parents fail to address the issue of socio-economic status or the potential role of genetic and family traits in pulmonary function capabilities.[210] Moreover, the reductions reported in the literature are small and of uncertain clinical or biological significance, and are contradicted by a number of studies which reportedly have observed no effect of parental smoking on children's lung function.

Sick Building Syndrome

Advocates of smoking restrictions in the workplace argue that ETS exposure gives rise to a number of complaints, including headaches, nausea, coughs, sore eyes and breathing difficulties. However, recent research indicates that this complex pattern of symptoms, the so-called "sick building syndrome," commonly occurs in modern office buildings whether or not smokers are present.[217-219]

Canadian researchers attending the 1983 University of Geneva symposium on ETS reported on their extensive review of over 150 indoor air quality evaluations of office buildings compiled by U.S. government agencies, universities and others.[218] After an examination of the data, the authors concluded that smoking did not significantly affect either indoor atmospheres or the frequency of worker complaints and symptoms:

> The review of available studies does not provide any objective evidence that either pollution levels or patterns of health related complaints differ in some remarkable way between locations with or without smoking restrictions.

They did observe that "inadequate" ventilation creates conditions "where discomfort and illness result irrespective of whether or not smoking is permitted." The researchers reaffirmed their findings in a report published in 1987.[220]

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Government investigators from Health and Welfare Canada (HWC), reporting on 94 building studies, noted that in only five percent of the cases were the complaints attributable to indoor constituents such as photocopy machine emissions and ETS.[221]

Similarly, in a review of 203 air quality investigations of government and business offices, schools and health care facilities by the U.S. National Institute of Occupational Safety and Health (NIOSH), U.S. government researchers concluded that tobacco smoke played a contributing role in generating complaints in only four of the buildings investigated.[217] A large majority of all complaints was traced to general building contamination and inadequate ventilation.

In 1988, a representative of a firm specializing in the maintenance of office air conditioning and heating systems reported on 223 individual indoor air quality investigations of publicly and privately owned office buildings.[219] As in the NIOSH investigations, ETS was involved in only four percent of the buildings investigated. He stated that the majority of indoor air quality problems in modern office buildings may be traced to inadequate fresh air circulation and to poorly maintained ventilation systems which act as breeding grounds for fungi, bacteria and other contaminants. He also suggested that visible

tobacco smoke ought to be considered a symptom, not a cause, of general indoor air quality problems, in that ETS is often the only visible sign that a ventilation problem exists.

It is perhaps understandable, given the easy recognition of ETS, that persons experiencing sick building symptoms may blame ETS as the cause. Indeed, Canadian researchers have verified that the mere visibility or presence of tobacco smoke may provoke claims that ETS is the cause of reported symptoms and complaints.[222] However, removal of ETS through smoking bans may serve only to divert attention from more basic, underlying indoor air quality problems. Indeed, as one commentator recently remarked: "Removing the smoker entirely, then, may not affect health and comfort problems in 95% to 98% of sick buildings."[223]

ENVIRONMENTAL TOBACCO SMOKE AND INDOOR AIR QUALITY

It is often suggested that ETS is a major source of indoor pollution. However, the scientific literature indicates that, except under experimental or other extraordinary conditions, ETS does not have a significant influence on the quality of indoor air.[224] The claim that ETS is a significant source of indoor air pollution (and, as such, a hazard to health) is based upon several unwarranted assumptions and misrepresentations of the scientific data. This section will provide an analysis of the ETS/indoor air quality question. The nature of ETS, its constituents, its contribution to indoor air, and the methods employed to measure exposure to it will be addressed.

Sidestream Smoke

It is frequently claimed that sidestream smoke, or the smoke from the burning end of the cigarette, contains much higher amounts of certain constituents than mainstream smoke, or the smoke inhaled by smokers. Such a claim implies that a nonsmoker has an increased risk of disease because of his exposure to sidestream smoke, which has more allegedly "toxic" or harmful constituents than smoke inhaled by the smoker. However, this argument is extremely misleading for several reasons, but most notably because it fails to take into account that sidestream smoke is immediately

diluted in the surrounding air. This diluted sidestream smoke is more accurately called "ETS."

The importance of dilution on sidestream smoke in room air cannot be overemphasized. Scientists have estimated that under normal, realistic conditions, the levels of major constituents of ETS are only fractions of those found in mainstream smoke.[225]

The potential toxicity of ETS has been assessed in a number of studies.[226-231] These studies typically tested the body fluids of nonsmokers exposed to ETS for mutagens, or substances capable of altering the genetic structure of cells. Although it can be argued that the scientific literature on this subject is very limited, and that the laboratory tests employed are crude, the studies nevertheless reported no mutagenic activity attributable to ETS exposure in the body fluids of nonsmokers.

Animal inhalation experiments using sidestream smoke or constituents of sidestream smoke are also inconclusive.[232,233-235] German scientists exposed rats and hamsters to very high levels of sidestream smoke during a 90-day inhalation experiment.[232] The researchers reported no significant physiological effects on the tissues of the animals. Researchers from the American Health Foundation reported similar results, i.e., no significant increase in lung tumors among animals exposed to sidestream smoke.[233-235]

In his comprehensive review of the literature on suspected pulmonary carcinogens, Dr. Domingo Aviado observed that none of the constituents in sidestream smoke which have been identified as potentially carcinogenic has induced pulmonary cancer in animals under experimental conditions.[236]

Constituents of ETS

A number of constituents have typically been cited in the literature for determining the contribution of ETS to the indoor air. These include carbon monoxide, particulates, nicotine, nitrosamines and others. However, the use of one or any combination of such constituents to determine ETS levels presents many problems. For example, although analytic and sampling methods continue to improve, there is at present no completely satisfactory and uniform procedure for measuring ETS.[237,238] Moreover, although the findings of studies which measure constituents in experimental conditions (e.g., in unventilated smog chambers) are cited to dramatize the potential effect of ETS on indoor air, they have little, if any, similarity to those studies which measure ETS in realistic settings. And finally, with the exception of nicotine, none of the constituents which have been used as substitute measures for ETS is characteristic of ETS alone. Other sources, such as heaters, stoves and furnishings, generate substances at levels greater than those found in ETS.

Exposure to ETS

Published studies indicate that nonsmoker exposure to ETS under normal, everyday conditions is minimal. For example, researchers report that there is little difference in ambient levels of carbon monoxide in smoking and nonsmoking areas of workplaces and public places.[220,239] Other studies indicate that ETS contributes less than half of the total particulates in the air of an average public place.[240-242] Nicotine often is used as a marker for ETS exposures because it is unique to tobacco smoke. Typical measurements of nicotine range from an exposure equivalent of 1/100 to 1/1000 of one filter cigarette per hour.[243-248] This means that a nonsmoker would have to spend from 100 to 1000 hours in an office, restaurant or public place in order to be exposed to the nicotine equivalent of a single cigarette.

It has been reported that cotinine, a substance converted from nicotine by the body, can be used to measure nonsmoker exposure to tobacco smoke.[249] While some reports appear to endorse the claim that cotinine is a reliable marker for total exposure to tobacco smoke,[250-253] others do not.[256-261] For example, researchers have reported that individuals metabolize nicotine in different ways at different times and that elimination rates for cotinine vary among individuals.[262-264] In addition, recent research indicates that

diet may contribute to levels of nicotine and cotinine found in the body.[265] Scientists have also noted that different methods of analysis may influence final recorded levels of cotinine.[266,267] Thus, scientific research casts serious doubt on the use of cotinine as a marker for total tobacco smoke exposure.

Summary

Even a cursory review of the published literature on ETS demonstrates that, at most, tobacco smoke has only a minor influence upon ordinary indoor environments compared to common household or workplace sources, such as cooking stoves, heaters and furnishings. Yet tobacco smoke, because it is visible, has become a target for those who apparently want a quick, simple solution to the problem of indoor air pollution. However, as one Canadian scientist observed, "the claim that smoking is responsible for indoor air pollution is an oversimplification of a complex, multi-source problem."[268]

ENVIRONMENTAL TOBACCO SMOKE AND THE PUBLIC

Governmental Regulations and Individual Rights

Although antismoking organizations claim to seek protection for the health and welfare of the nonsmoker, their efforts to make smoking socially unacceptable are part of a broader objective -- the severe restriction or even prohibition of smoking by governmental legislation. Of course, such regulations restrict the freedom of the smoker, and antismokers are compelled to justify this kind of political control of individual behavior. They have accomplished this, in large measure, by exploitation of the so-called "health" argument involving ETS exposures to nonsmokers.

For example, the most recent U.S. Surgeon General's Report on this subject (1986) contends that ETS causes lung cancer in adult nonsmokers and respiratory conditions in children, and that separation of smokers and nonsmokers is not effective in minimizing the nonsmoker's exposure to ETS.[5] The Report concludes that smoking bans will not only reduce ETS exposures, but will "alter smoking behavior and public attitudes about tobacco use." The Report further suggests that "over time, this may contribute to a reduction of smoking." Thus, the underlying motivation for the use of the ETS/health argument is to attain a "smoke-free society by the year 2000."

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The Surgeon General's tactics, however, have been questioned by a number of critics. One reviewer suggested that the Surgeon General's conclusions were based on "flimsy" evidence presented in an effort to "divert attention" from important health concerns such as the "poisoning of the environment."[269] A U.S. Congressman, in a letter to the Congressional Record, wrote that "the conclusions in the Surgeon General's Report are not supported by the research in his own report."[270] Moreover, scientific studies aboard commercial aircraft and in offices indicate, contrary to the Surgeon General's Report, that simple separation of smokers and nonsmokers effectively minimizes ETS exposures for nonsmokers.[271-273]

Five U.S. labor union presidents criticized the Surgeon General's Report for minimizing the risk imposed by workplace toxins, for shifting the burden of cleaning up the workplace to workers, and for dismissing the importance of engineering controls (e.g., ventilation) in the maintenance of a safe and comfortable workplace.[274] The Tobacco Institute, in a comprehensive review of the issue, observed that premature scientific conclusions about ETS, together with increasing political pressure to accept the Surgeon General's conclusions, have "brought scientific integrity to a crossroads."[275]

An Australian writer recently summarized the issue of governmental influence on the ETS question.[276] He wrote:

> Those involved in government anti-smoking activities should be aware of the tenuous nature of the data on passive smoking and health effects. The deliberate use of fear inducing tactics by misrepresenting such data by dubious extrapolations is propaganda and not health education.

It is clear that once the health claims regarding environmental tobacco smoke are placed in their proper scientific perspective, the issue of smoking in public places becomes a social and political one. As a former director of the WHO wrote recently, "the question as to whether the conceivable theoretical possibility of a risk calls for official preventive measures is not a medical but a politico-social one."[277]

The issue thus involves whether or not public policy should mandate prohibition of smoking because it is seen by some as a nuisance or annoyance. If the decision is reached in favor of regulation, then one must decide whether the same line of reasoning applies to other kinds of "annoyances" encountered in everyday life and whether those "annoyances" should also be banned. The implication of such reasoning should be obvious to anyone who is opposed to unwarranted governmental intrusion into people's lives.

Thus, a German scientist recently concluded:

> Whether a real risk is involved which in the future can be demonstrated on the basis of measurable results remains an open question. In light of this health officials should concentrate on more significant environmental problems in the long term interest of society rather than wasting time and money on trivial issues.[278]

What is the Solution?

An article on the principles of public policy relating to smoking suggests an alternative to governmental regulations. According to this article, "the appropriate form of public policy is the promotion of courtesy and cooperation between smokers and nonsmokers, rather than outright prohibition."[279] This solution relies upon common sense, courtesy and the free marketplace for decisions concerning smoking. More importantly, however, this solution avoids unnecessary governmental interference with the subsequent loss of individual freedom.

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